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Predictors of left atrial strain recovery in patients hospitalized with acute heart failure with reduced ejection fraction

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ABSTRACT

Aims: Left atrial (LA) function is crucial in heart failure (HF) pathophysiology, and its impairment is associated with adverse outcomes. LA reservoir strain (LASr), assessed via speckle-tracking echocardiography, has emerged as a sensitive marker of LA mechanics, yet its recovery during acute HF remains unclear. This study aimed to identify the clinical and echocardiographic predictors of LASr improvement in patients hospitalized with acute decompensated HF with reduced ejection fraction (HFrEF).

Methods: This retrospective study included 63 hospitalized patients with acute decompensated HFrEF (LVEF <40%). Patients were classified into improvers (≥15% increase in LASr) and non-improvers based on LASr recovery during hospitalization. Clinical and echocardiographic parameters were compared between groups, and independent predictors of LASr improvement were identified through logistic regression analysis. Model performance was evaluated using ROC and decision curve analyses.

Results: LASr improved in 38% of patients (improvers: n=24), increasing from 7.8% (IQR: 4.8–11.5) to 10.0% (IQR: 7.0–13.0, p=0.035). Compared to non-improvers, improvers had higher LVEF (p=0.009), smaller LV end-diastolic diameter (p=0.015), and lower prevalence of moderate-to-severe mitral regurgitation (p=0.012). In multivariate analysis, LVEF (OR: 1.204, 95% CI: 1.040–1.395) and LV end-diastolic diameter (OR: 0.879, 95% CI: 0.780–0.990) predicted LASr recovery, while moderate-to-severe MR was associated with lower recovery (OR: 0.170, 95% CI: 0.029–0.988). ROC analysis confirmed model performance (AUC: LVEF 0.852, EDD 0.831, MR 0.779).

Conclusion: LASr improvement during hospitalization is closely linked to baseline LV function, ventricular dimensions, and MR severity, highlighting its dynamic nature in acute HF and potential as a marker of cardiac recovery.

Keywords: Left atrial reservoir strain, heart failure with reduced ejection fraction, acute heart failure, strain recovery, echocardiographic assessment

INTRODUCTION

The left atrium (LA) plays a crucial role in cardiovascular hemodynamics by regulating left ventricular (LV) filling and adapting to changing circulatory demands.[1] However, in heart failure (HF), increased LV filling pressures and structural remodeling impair LA function, contributing to pulmonary congestion and worsening symptoms.[2] Conventional volumetric assessments may not fully capture these functional impairments, highlighting the need for more refined imaging techniques.[3]

Speckle-tracking echocardiography (STE)-derived LA strain (LAS) has emerged as a valuable tool for assessing LA function beyond traditional measurements.[4] Among its components, reservoir strain is particularly relevant, as it reflects LA distensibility and compliance during ventricular systole, integrating both atrial and ventricular interactions for a more comprehensive assessment of LA function.[5] Reduced LAS has been linked to adverse outcomes, including higher rates of hospitalization and mortality, independent of LV function.[3] While LAS changes during hospitalization may provide insights into treatment response, its dynamic trajectory remains incompletely understood.

Although LAS improves in some patients following decongestive therapy, others exhibit persistent dysfunction despite volume optimization.[4] Given its prognostic significance, identifying the clinical and echocardiographic predictors of LAS recovery in acute heart failure (AHF) is

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essential. However, this area remains largely unexplored. In this study, we aimed to investigate the determinants of LAS improvement in patients with AHF and reduced ejection fraction (HFrEF).

METHODS

Ethics

The study was conducted in accordance with the Declaration of Helsinki and this study was initiated with the approval of the Clinical Researches Ethics Committee of Başakşehir Çam and Sakura City Hospital (Date: 27.04.2022, Decision No: 135). Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Study Population

This retrospective study included patients presenting to the emergency department (ED) with AHF and a reduced ejection fraction (EF <40%), who were subsequently hospitalized in the cardiology ward. AHF was defined according to current guidelines as the rapid or progressive onset of symptoms and/or signs of HF, severe enough to necessitate urgent medical evaluation, resulting in unplanned hospital admission or an ED visit.[6]

Patients were excluded if they met any of the following criteria: delayed admission to the cardiology ward (>24 hours from ED presentation to ensure consistency in the timing of echocardiographic evaluation following early diuretic administration and to avoid variability in volume status that could affect strain measurements.), recent acute coronary syndrome (<1 month), requirement for inotropic support during hospitalization, presence of primary valvular heart disease or prior mitral valve interventions, suboptimal imaging quality insufficient for speckle-tracking echocardiographic analysis, or advanced chronic kidney disease (CKD) defined as an estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m^2 or dependence on dialysis.

Transthoracic echocardiography was performed 2 to 4 hours after intravenous diuretic administration to ensure that imaging was conducted before significant hemodynamic alterations in LA function occurred, while avoiding delays in patient management. Discharge decisions were made based on clinical stability, resolution of congestion symptoms, and improvement in standard HF parameters, as per institutional HF management protocols. Patients' demographic data, laboratory parameters, and echocardiographic measurements were systematically recorded.

Echocardiographic Examination

Transthoracic echocardiographic (TTE) evaluations were performed using an EPIQ CVx (Philips, Netherlands) ultrasound system equipped with an S5-1 phased-array transducer. Measurements of LV and LA dimensions, LV ejection fraction (LVEF), and diastolic LV filling velocities were obtained in accordance with the recommendations of the European Association of Cardiovascular Imaging (EACVI) and the American Society of Echocardiography (ASE).[7] Right ventricular (RV) systolic function was assessed using tricuspid annular plane systolic excursion (TAPSE) and RV systolic myocardial velocity (RVSm), while systolic pulmonary arterial pressure (sPAP) was estimated based on the tricuspid regurgitation (TR) velocity. LV and LA volumes were calculated using the biplane Simpson's method and the LA volume index (LAVi) was obtained by indexing LA volume to body surface area (BSA). Mitral and TR severity was assessed according to current echocardiographic guidelines.[8]

Strain Analysis

LA strain (LAS) was assessed using two-dimensional speckle-tracking echocardiography (2D-STE) in accordance with current guidelines.[9] Apical four-chamber (A4C) and two-chamber (A2C) views were acquired, and the average was used to enhance reproducibility. The endocardial border was automatically traced with manual adjustments as needed, ensuring optimal tracking. Frame rates were set between 50–70 frames per second, and offline analyses were performed using QLAB software (Philips, Netherlands).

LAS components included reservoir strain (LASr), reflecting LA expansion during LV systole, conduit strain (LAScd), representing passive emptying in early diastole, and contraction strain (LASct), corresponding to active contraction in late diastole. In atrial fibrillation (AF) patients, only LASr was analyzed due to the absence of atrial contraction. A representative LA strain analysis is shown in **Figure 1**.

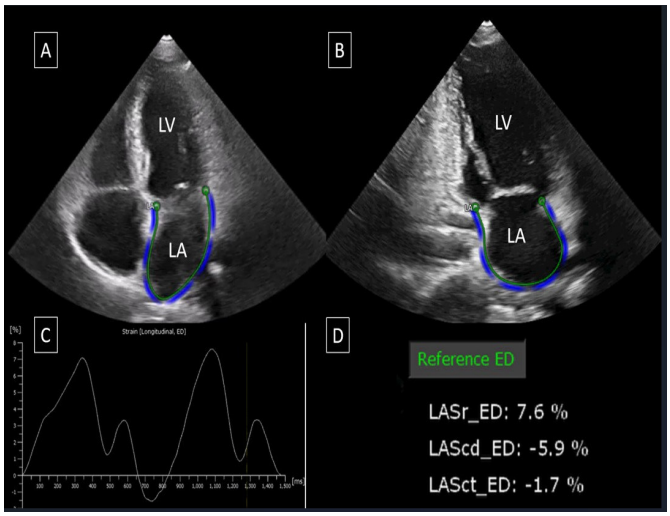

Figure 1. Left atrial strain assessment using speckle-tracking echocardiography (**A**) Apical four-chamber (A4C) and (**B**) apical two-chamber (A2C) views demonstrating left atrial (LA) strain analysis. The endocardial border is manually traced, and strain curves are generated. (**C**) Strain-time curve showing longitudinal strain measurements of the left atrium. (**D**) Strain values at end-diastole (ED), including LA reservoir strain (LASr_ED), conduit strain (LAScd_ED), and contractile strain (LASct_ED), are displayed

For LV global longitudinal strain (LV-GLS), A4C, A2C, and apical long-axis (APLAX) views were analyzed. RV and right atrial (RA) strain were obtained from an RV-focused A4C and optimized A4C view, respectively. As with LA strain, only RASr was analyzed in AF patients.

Statistical Analysis

Patients were classified as improvers (≥15% increase in LASr from admission to discharge) or non-improvers, based on previously established thresholds.[10] Group comparisons for

demographic, laboratory, and echocardiographic parameters were conducted. Continuous variables were assessed for normality using the Shapiro-Wilk test; normally distributed data were reported as mean±standard deviation (SD) and compared using the independent Samples t-test, while non-normally distributed data were presented as median (IQR) and analyzed using the Mann-Whitney U test. Categorical variables were expressed as n (%) and compared using the Chi-square or Fisher's exact test.

Univariate and multivariate logistic regression analyses were performed to identify predictors of LASr recovery. Variables with p<0.25 in univariate analysis and those deemed clinically relevant were included in the multivariate model. Model performance was assessed using the Hosmer-Lemeshow test. Multicollinearity among independent variables was assessed using the variance inflation factor (VIF), and all variables included in the final multivariate model had VIF values <2, indicating no significant collinearity. The association between LASr improvement and continuous variables was evaluated using Spearman or Pearson correlation, as appropriate.

Receiver operating characteristic (ROC) analysis was used to determine cutoff values, area under the curve (AUC), sensitivity, and specificity for significant predictors. Model performance was further assessed with decision curve analysis (DCA) and calibration plots.

To ensure measurement reliability, interobserver and intraobserver variability of strain analyses was evaluated in a randomly selected subset of 15 patients, quantified using intraclass correlation coefficients (ICC).

Statistical analyses were performed using SPSS version 30 (IBM, Armonk, NY, USA) and R version 4.4.2, with p<0.05 considered statistically significant.

RESULTS

A total of 72 patients were initially screened, and after applying the exclusion criteria, 63 patients were included in the final analysis. The median age of the study population was 68 years (IQR: 58–76), and 40 patients (63%) were male. The median LVEF was 33.5% (IQR: 29.8–38.3). Ischemic cardiomyopathy was the predominant etiology, observed in 73% of patients. The prevalence of coronary artery disease (CAD) was 83%, while diabetes mellitus (DM) and hypertension (HT) were present in 49% and 68% of patients, respectively. At admission, 44 patients (70%) were classified as NYHA class IV. Although not statistically significant, hypertension (p=0.054) and CKD (p=0.074) were more prevalent among non-improvers, indicating a possible trend toward association with limited LASr recovery. Baseline demographic, clinical and echocardiographic characteristics of the study population are summarized in **Table 1, 2**.

Decongestive therapy resulted in significant reductions in E/e' ratio, TR velocity, and sPAP. The E/e' ratio decreased from a median of 18.6 (IQR: 13.1–19.8) at admission to 12.2 (IQR: 11.7–12.9) at discharge (p=0.017). TR velocity reduced from 2.4 m/s (IQR: 2.1–3.2) to 2.2 m/s (IQR: 2.0–2.6) (p=0.041). sPAP

Table 1. Baseline and echocardiographic characteristics of non-improvers and improvers

Variable	Non-improver (n=35)	Improver (n=28)	Total (n=63)	p-value*
Baseline demographics				
Age, years	63 (55.5-74)	70.5 (63.75-78)	68 (58-76)	0.231
Male, n(%)	23 (66%)	17 (61%)	40 (63%)	0.682
CAD, n(%)	29 (83%)	23 (82%)	52 (83%)	0.941
DM, n(%)	19 (54%)	12 (43%)	31 (49%)	0.311
HL, n(%)	15 (43%)	8 (29%)	23 (37%)	0.301
HT, n(%)	27 (77%)	16 (57%)	43 (68%)	0.054
CMP type n(%)				0.854
Dilated	8 (23%)	6 (21%)	14 (22%)	
Ischemic	25 (71%)	21 (75%)	46 (73%)	
History of stroke, n(%)	4 (11%)	2 (7%)	6 (10%)	0.545
CKD, n(%)	16 (46%)	7 (25%)	23 (37%)	0.074
AF, n(%)	15 (43%)	8 (29%)	23 (37%)	0.211
COPD, n(%)	5 (14%)	4 (14%)	9 (14%)	0.982
ICD, n(%)	7 (20%)	6 (21%)	13 (21%)	0.864
Systolic BP, mmHg	117.5 (110.2-124.9)	118.9 (111.3-125.7)	118.2 (110.8-125.3)	0.617
HR, bpm	76 (70-88)	85 (74-97)	78 (70-91)	0.139
BMI, kg/m²	26.81 (22.9-32.2)	29.3 (29.3-29.3)	29.3 (23.5-31.3)	1.000
Hospitalization duration, days	7 (5-8)	6 (4.25-8)	7 (5-8)	0.118
NYHA, n(%)				0.789
Class 3	11 (31%)	8 (29%)	19 (30%)	
Class 4	24 (69%)	20 (71%)	44 (70%)	
Heart failure medications				
Loop diuretic, n (%)	34 (97%)	27 (96%)	55 (87%)	0.874
Thiazide, n (%)	9 (26%)	6 (21%)	15 (24%)	0.653
Beta-blocker, n(%)	30 (86%)	21 (75%)	51 (81%)	0.156
ACEi/ARB, n(%)	28 (80%)	23 (82%)	51 (81%)	0.875
MRA, n(%)	12 (34%)	13 (46%)	25 (40%)	0.254
SGLT-2 inhibitor	14 (40%)	11 (39%	25 (40%)	0.812
ARNI	8 (23%)	6 (21%)	14 (22%)	0.729
Laboratory findings				
Creatinine, mg/dl	1.4 (1.0-1.8)	1.4 (1.1-1.6)	1.4 (1.0-1.7)	0.606
eGFR, ml/min/1.73m²	46 (31.7-71.5)	47 (35.5-59.5)	47 (34.5-64)	0.527
HB, g/dl	11.4 (9.9-13.8)	11.7 (10.6-13.5)	11.6 (10.4-13.5)	0.571
Admission BNP, pg/ml	10880.5 (5635.2-21062)	8761 (4042.2-17219.2)	9884 (4285-17376)	0.191
Discharge BNP pg/ml	8065 (3512.5-13294.7)	5715 (3464-10255)	6850 (3820-12792.2)	0.768

* Continuous variables are presented as median (interquartile range), and categorical variables are expressed as n (%). A p-value < 0.05 was considered statistically significant. Abbreviations: AF: Atrial fibrillation, ARNI: Angiotensin receptor-neprilysin inhibitor, BMI: Body-mass index (kg/m²), BNP: B-type natriuretic peptide (pg/ml) CAD: Coronary artery disease, CKD: Chronic kidney disease, CMP type: Cardiomyopathy type, COPD: Chronic obstructive pulmonary disease, Diastolic BP: Diastolic blood pressure (mmHg), DM: Diabetes mellitus, eGFR: Estimated glomerular filtration rate (ml/min/1.73m²), HB: Hemoglobin (g/dl), HL: Hyperlipidemia, HR: Heart rate (bpm), HT: Hypertension, ICD: Implantable cardioverter defibrillator, MRA: Mineralocorticoid receptor antagonist, NYHA: New York Heart Association functional classification, SGLT2: Sodium-glucose cotransporter-2

Table 2. Comparison of echocardiographic parameters between non-improvers and improvers at admission and discharge

Variable	Non-Improver (n=35)	Improver (n=28)	Total (n=63)	p-value*
Admission				
Left ventricle				
EDD, mm	60 (57-63)	56 (53-59)	58 (55-61)	0.014
ESD, mm	50 (47-53)	46 (43-48)	48 (45-51)	0.046
EF, %	32 (26-35)	38 (32-39)	33.5 (29.75-38.25)	0.004
E/e'	12 (11.5-12.8)	12.9 (12.1-13.75)	12.1 (11.6- 12.8)	0.061
TR vel, cm/s	2.38 (2.1-3.2)	2.5 (2.2-3)	2.38 (2.1-3.2)	0.715
SPAP, mmHg	25 (25-27)	25.3 (25-27.1)	25 (25-27)	0.833
TAPSE, mm	17 (12-20)	17 (16-18)	17 (12-19)	0.752
RVSM, mm	9 (7-10)	9.7 (8.5-12.5)	9.1 (7.75-10)	0.906
LA (A-P), mm	43 (40-46)	42 (39-45)	42.5 (39.5-45.5)	0.782
LA Volume, ml	92 (80-129)	84 (72-104)	93.5 (75-127)	0.170
LAVI, mL/m²	51.1 (44.4-71.6)	46.6 (40-57.7)	51.9 (41.6- 70.5)	0.170
Mitral regurgitation, n (%)				0.032
None	2 (6%)	5 (18%)	7 (11%)	
Mild	6 (17%)	12 (43%)	18 (29%)	
Moderate	16 (46%)	7 (25%)	23 (37%)	
Severe	11 (31%)	4 (14%)	15 (24%)	
Tricuspit regurgitation, n (%)				0.512
Mild	8 (23%)	8 (29%)	23 (37%)	
Moderate	19 (54%)	14 (50%)	28 (44%)	
Severe	8 (23%)	6 (21%)	12 (19%)	
Strain parameters				
LASr, %	5.6 (4.2-10.3)	8.2 (6.9-13.0)	7.8 (4.8-11.5)	0.004
LAScd, % **	-6.1 (-9.3--3.9)	-5.7 (-7.3--3.3)	-5.8 (-8--3.3)	0.449
LASct, %	-3.6 (-5.8--1)	-1.1 (-2-0)	-1.7 (-5.15--0.4)	0.023
RASr, %	9.9 (6.2-17)	12.2 (9.6-16)	11.2 (6.3-16)	0.447
RAScd, %	-8.2 (-10.4--4.6)	-7.0 (-10.7--4.15)	-7.9 (-10.4--4.3)	0.643
RASct, %	-3.8 (-9.6--1.7)	-6.15 (-11.4--1.4)	-4.1 (-9.9--1.45)	0.597
RVFW, %	-12.6 (-17--6.9)	-14.1 (-14.2--8.6)	-12.6 (-14.2--8.6)	0.971
RV4C, %	-10.6 (-10.6--7.5)	-9.3 (-10.1--6.6)	-10.1 (-10.6--7.5)	0.409
LV, %	-10.7 (-12.2--8.7)	-12.7 (-15.8--9.2)	-11.2 (-12.5--9)	0.046
Discharge				
Left ventricle				
EDD, mm	58 (55-62)	55 (52-59)	57 (54-61)	0.031
ESD, mm	49 (46-52)	44 (41-47)	46 (42-49)	0.046
EF, %	34 (26-40)	40 (36-44)	36 (33-38)	0.015
LA, mm	42.8 (39.7-45.8)	41 (38.5-44)	42.5 (41-44)	0.083
E/e'	18.5 (13.1-19.8)	18.5 (18.5-18.5)	18.5 (13.1-19.8)	0.900
TR vel, cm/s	2.6 (2.6-2.6)	2.5 (2.3-2.7)	2.6 (2.3-2.7)	1.000
SPAP, mmHg	32 (32-32)	31.5 (30-33)	32 (30-33)	1.000
TAPSE, mm	15 (12-21)	22 (22-22)	15 (12-21)	0.194
RVSM, mm	9.9 (7-9.95)	11.3 (11-11.6)	9.9 (7-11)	0.004
LA volume, ml	94 (81-120)	111 (97.75-120)	106 (87-120)	0.084
LAVI, ml/m²	52.7 (45-66.6)	61.6 (55.2-66.6)	58.8 (48.3-66.6)	0.084
Mitral regurgitation, n (%)				0.044
None	6 (17%)	8 (29%)	14 (22%)	

The table continues

Table 2. Comparison of echocardiographic parameters between non-improvers and improvers at admission and discharge (continues)

Mild	18 (51%)	12 (43%)	30 (48%)	
Moderate	9 (26%)	8 (29%)	17 (27%)	
Severe	2 (6%)	0	2 (3%)	
Tricuspit regurgitation, n (%)				
Mild	19 (54%)	21 (75%)	40 (63%)	0.221
Moderate	10 (29%)	5 (18%)	15 (24%)	
Severe	6 (17%)	2 (7%)	8 (13%)	
Strain parameters				
LASr, %	6.2 (4.5-10.8)	12 (7.5-18.1)	8.3 (5.9-14.4)	0.001
LAScd, %	-5.1 (-9.3--3.6)	-6.7 (-7.8--4.6)	-6.7 (-9.2--4)	0.073
LASct, %	-1.3 (-4.2--0.6)	-3.9 (-9.1--0.9)	-1.8 (-5.6--0.8)	0.009
RASr, %	7.85 (4.9-14.8)	13.8 (7.9-22.8)	11.6 (5.4-20.1)	0.088
RAScd, %	-4.9 (-5.6--2.5)	-5.6 (-8.7--3.7)	-5 (-7.7--3.05)	0.152
RASct, %	-3.4 (-8.05--1.0)	-6.4 (-11.4--4.7)	-5.5 (-9.3--2.4)	0.088
RVFW, %	-9.8 (-11.6--5.6)	-17.1 (-21--13.2)	-11.6 (-13.2--9.8)	0.007
RV4C, %	-7.6 (-9.6--3.8)	-16.3 (-22--10.6)	-9.6 (-10.6--7.6)	0.007
LV, %	-11.5 (-12--8.8)	-13.1 (-17--9)	-11.7% (-13.0-9.5)	0.025

* Continuous variables are presented as median (interquartile range), and categorical variables are expressed as n (%). A p-value < 0.05 was considered statistically significant. **LAScd, LASct, RAScd and RASct values are reported only for patients in sinus rhythm. Abbreviations: EDD: End-diastolic diameter (mm), EF: Ejection fraction (%), ESD: End-systolic diameter (mm), LA (A-P): Left Atrium antero-posterior (mm), LAScd: Left atrial strain (Conduit) (%), LASct: Left atrial strain (contraction) (%), LASr: Left atrial strain (reservoir) (%), LAVI: Left atrial volume index (ml/m^2), LA volume: Left atrial volume (ml), LV: Left ventricle (mm), RAScd: Right atrial strain (conduit) (%), RASct: Right atrial strain (contraction) (%), RASr: Right atrial strain (reservoir) (%), RV4c: Right ventricular 4C strain (%), RVFW: Right ventricular free wall strain (%), RVSM: Right ventricular systolic motion (mm), SPAP: Systolic pulmonary artery pressure (mmHg), Systolic BP: Systolic blood pressure (mmHg), TAPSE: Tricuspid annular plane systolic excursion (mm), TR: Tricuspid regurgitation, TR vel: Tricuspid regurgitation velocity (cm/s)

declined from 25.0 mmHg (IQR: 25.0–27.0) to 22.5 mmHg (IQR: 21.0–24.0) (p=0.037). LASr improved significantly, increasing from 7.8% (IQR: 4.8–11.5) at admission to 10.0% (IQR: 7.0–13.0) at discharge (p=0.035). Mitral regurgitation (MR) severity decreased significantly (p=0.011), with the proportion of patients with no MR increasing from 11% to 22% and those with severe MR decreasing from 24% to 3%. These findings are presented in detail in **Table 3**.

Univariate analysis identified several parameters associated with LASr recovery, which were further assessed in the multivariate logistic regression model. EF was found to be an independent predictor of recovery (OR: 1.204, 95% CI: 1.040–1.395, p=0.013), while left ventricle end-diastolic diameter (EDD) was inversely associated (OR: 0.879, 95% CI: 0.780–0.990, p=0.034). Moderate-to-severe MR also demonstrated a significant negative predictive value (OR: 0.170, 95% CI: 0.029–0.988, p=0.048). The regression results are summarized in **Table 4**. The graphical representation of the model is shown in **Figure 2A**, while the decision curve analysis (**Figure 2B**) demonstrated its clinical utility across a range of risk thresholds. The calibration plot (**Figure 2C**) confirmed good model performance, further supported by the Hosmer-Lemeshow test (Chi-square=12.82, df=8, p=0.118). The model demonstrated a sensitivity of 82% and a specificity of 86% and demonstrated good overall performance, with a Nagelkerke R^2 of 0.698 and a Cox & Snell R^2 of 0.490. Model calibration was acceptable based on the non-significant Hosmer–Lemeshow test (χ^2=11.287, df=8, p=0.186), and the -2 log likelihood value was 44.101.

ROC analysis was conducted to assess the predictive performance of EF, reversed EDD, and none-mild MR for LASr recovery. MR was categorized as none-mild versus moderate-severe to ensure a clinically meaningful distinction between patients with minimal versus significant volume overload. Since larger EDD values were associated with a lower probability of recovery, EDD values were reversed to maintain consistency in AUC interpretation. The analysis yielded an AUC of 0.852 (95% CI: 0.752–0.951) for EF, 0.831 (95% CI: 0.725–0.936) for reversed EDD, and 0.779 (95% CI: 0.659–0.898) for None-Mild MR (**Figure 3A**). Restricted cubic spline plots were used to illustrate the association between EF, EDD, and the probability of LASr recovery (**Figure 3B, 3C**). EF showed a positive association with recovery probability, while EDD demonstrated a non-linear relationship, with lower values being linked to higher recovery probability, followed by a plateau at larger values.

For reliability assessment, interobserver and intraobserver variability of echocardiographic parameters, including strain measurements, were evaluated in a subset of 15 patients. The ICC were 0.89 (95% CI: 0.82–0.94) for interobserver variability and 0.93 (95% CI: 0.88–0.96) for intraobserver variability, demonstrating good overall agreement.

DISCUSSION

The present study demonstrated that in patients hospitalized with AHF and HFrEF, LASr significantly improved following decongestive therapy, highlighting the responsiveness of LA mechanics to volume optimization, and this improvement was independently predicted by higher baseline EF, smaller LV dimensions, and less severe MR assessed at hospital admission. As the first study to specifically investigate LA strain recovery in this population, our findings emphasize that LASr recovery during hospitalization reflects not only acute decongestion but also underlying cardiac structure and valvular function.

Table 3. Changes in echocardiographic and strain parameters from admission to discharge

Parameter	Admission	Discharge	p-value
EDD, mm	58 (55-61)	57 (54-61)	0.679
ESD, mm	48 (45-51)	47 (44-50)	0.138
EF, %	34 (29.7-38.2)	35 (31-39)	0.102
LA, mm	42.5 (39.5-45.5)	42.5 (41-44)	0.159
E/e'	18.5 (13.1-19.8)	12.1 (11.6-12.8)	0.017
TR velosity,	2.38 (2.1-3.2)	2.2 (2-2.6)	0.041
SPAP, mmHg	25 (25-27)	22.5 (21-24)	0.037
TAPSE, mm	17 (12-19)	15 (12-21)	0.208
RVSM	9.1 (7.7-10)	9.9 (7-11)	0.679
LAVI	51.9 (41.6-70.5)	58.8 (48.3-66.6)	0.455
Strain parameters			
LASr, %	7.8 (4.8-11.5)	10 (7-13)	0.035
LAScd, %	-5.85 (-8--3.3)	-6.7 (-9.2--4)	0.935
LASct, %	-1.7 (-5.1--0.4)	-1.8 (-5.6--0.8)	0.058
RASr, %	11.2 (6.3-16)	11.6 (5.4-20.1)	0.970
RAScd, %	-7.9 (-10.4--4.35)	-5 (-7.7--3.05)	0.236
RASct, %	-4.1 (-9.9--1.45)	-5.5 (-9.3--2.4)	0.922
RVFW, %	-12.6 (-14.2--8.6)	-11.6 (-13.2--9.8)	0.172
RV4C, %	-10.1 (-10.6--7.5)	-9.6 (-10.6--7.6)	0.172
LV, %	-11.2 (-12.5--9)	-11.7% (-13.0 -9.5)	0.057
Mitral regurgitation, n (%)			0.011
None	7 (11%)	14 (22%)	
Mild	18 (29%)	30 (48%)	
Moderate	23 (37%)	17 (27%)	
Severe	15 (24%)	2 (3%)	
Tricuspid regurgitation, n (%)			0.221
Mild	23 (37%)	40 (63%)	
Moderate	28 (44%)	15 (24%)	
Severe	12 (19%)	8 (13%)	

Continuous variables are presented as median (interquartile range), and categorical variables are expressed as n (%). A p-value < 0.05 was considered statistically significant. Abbreviations: EDD: End-diastolic diameter (mm), EF: Ejection fraction (%), ESD: End-systolic diameter (mm), LA (A-P): Left Atrium antero-posterior (mm), LAScd: Left atrial strain (Conduit) (%), LASct: Left atrial strain (contraction) (%), LASr: Left atrial strain (reservoir) (%), LAVI: Left atrial volume index (ml/m^2), LA volume: Left atrial volume (ml), LV: Left ventricle (mm), RAScd: Right atrial strain (conduit) (%), RASct: Right atrial strain (contraction) (%), RASr: Right atrial strain (reservoir) (%), RV4c: Right ventricular 4C strain (%), RVFW: Right ventricular free wall strain (%), RVSM: Right ventricular systolic motion (mm), SPAP: Systolic pulmonary artery pressure (mmHg), Systolic BP: Systolic blood pressure (mmHg), TAPSE: Tricuspid annular plane systolic excursion (mm), TR: Tricuspid regurgitation, TR vel: Tricuspid regurgitation velocity (cm/s)

LAS assessed by 2D-STE has emerged as a sensitive marker of atrial structural remodeling and functional impairment, providing insights beyond conventional echocardiographic parameters.[11] LAS is closely coupled with ventricular function throughout the cardiac cycle, reflecting the dynamic interplay between atrial compliance, ventricular filling pressures, and ventricular longitudinal shortening.[1] Although LA mechanics include reservoir, conduit, and contraction strains, we focused on reservoir strain due to its reliability across all patients, including those with AF, where other strain components cannot be accurately assessed.[12]

LAS has increasingly gained attention as a sensitive marker of cardiac hemodynamics and therapeutic response in patients with AHF.[4] Previous studies have evaluated the dynamics of LAS in diverse patient populations and HF phenotypes. Barki et al.[13] demonstrated that improvement in LAS following decongestion was strongly associated with better clinical outcomes, including reduced hospitalization rates, across patients with different EF. Similarly, Park et al.[3] highlighted LAS as a robust predictor of prognosis in AHF. Deferm et al.[4] further demonstrated that LASr improved from 6.4% to 8.8% during hospitalization and continued to rise to 13.4% at 6 weeks (p<0.001), emphasizing its role as a marker of treatment response. While their study provided valuable insights into the time course of LAS recovery, our study, which included a larger cohort, demonstrated that significant LASr improvement occurs even within the hospitalization period. Unlike prior studies with mixed HF phenotypes, we focused exclusively on HFrEF patients and assessed LA mechanics solely through reservoir strain, ensuring a consistent and rhythm-independent evaluation despite the relatively high prevalence of AF.

A novel finding of our study was that baseline cardiac structure significantly influenced LAS improvement. Specifically, patients with a higher baseline EF and smaller LV dimensions exhibited a greater magnitude of improvement in LAS after decongestive therapy. This may be explained by the fact that patients with relatively preserved LV function and less adverse cardiac remodeling at baseline have better myocardial reserve, allowing more complete recovery of LA mechanics after alleviating congestion.[14]

Our findings highlight baseline MR severity as an essential determinant of LAS improvement following treatment in AHF. Patients with less severe MR experienced more pronounced recovery in LA mechanics, supporting the notion that ongoing volume overload associated with significant MR imposes sustained mechanical stress on the LA, impairing its capacity for functional restoration despite adequate decongestion.[10] Interestingly, among patients with lower MR severity, those with higher baseline LASr values exhibited greater improvement. This suggests that preserved atrial mechanics may allow for a more dynamic recovery, whereas severely impaired LA function, potentially reflecting advanced structural remodeling, may limit the extent of reversibility. This aligns with prior studies suggesting that chronic MR adversely affects LA remodeling and compliance, ultimately limiting the potential for atrial functional recovery.[15] Additionally, in our cohort, RV strain improvement was more pronounced in patients with significant LA strain recovery. This suggests that enhanced LA function may contribute to better pulmonary venous unloading, reducing RV afterload and facilitating improved RV performance.[16] This compensatory response may reflect a more effective hemodynamic adaptation to decongestive therapy, warranting further investigation.

Given its sensitivity to hemodynamic changes and its association with structural remodeling, LASr recovery may serve as a valuable marker for assessing therapeutic response and identifying patients at risk of persistent atrial dysfunction despite decongestive treatment. Our findings suggest that beyond simply reflecting volume reduction, LASr improvement integrates information on baseline ventricular function and valvular integrity, which could have important

Table 4. Univariate and multivariate logistic regression analysis for predictors of left atrial reservoir strain recovery

	Univariate analysis				Multivariate analysis			
	p-value	OR	95% CI		p-value	OR	95% CI	
			Lower	Upper			Lower	Upper
Age, years	0.061	1.046	0.998	1.097	0.259	1.043	0.970	1.121
Male	0.621	0.754	0.246	2.312				
Diabetes mellitus	0.608	0.736	0.229	2.371				
Hypertension,	0.105	0.350	0.099	1.243	0.351	0.387	0.053	2.844
Atrial fibrillation	0.167	0.426	0.127	1.430	0.266	0.356	0.058	2.196
Systolic BP, mmHg	0.261	0.968	0.916	1.023				
eGFR, ml/min/1.73 m²	0.973	1.000	0.975	1.024				
BNP, pg/ml	0.246	1.000	1.000	1.000	0.980	1.000	1.000	1.000
End-diastolic diameter, mm	0.122	1.080	0.980	1.190	0.034	0.879	0.780	0.990
Ejection fraction, %	0.004	1.158	1.049	1.279	0.013	1.204	1.040	1.395
Left atrial volume index, ml/m²	0.305	0.977	0.933	1.022				
TAPSE, mm	0.991	1.001	0.787	1.274				
Systolic pulmonary artery pressure, mmHg	0.229	1.029	0.982	1.078				
Moderate-severe mitral regurgitation, %	<0.001	0.081	0.024	0.268	0.048	0.170	0.029	0.988

OR: Odds ratio, CI: Confidence interval, Systolic BP: Systolic blood pressure (mmHg), eGFR: Estimated glomerular filtration rate, BNP: Brain natriuretic peptide, TAPSE: Tricuspid annular plane systolic excursion

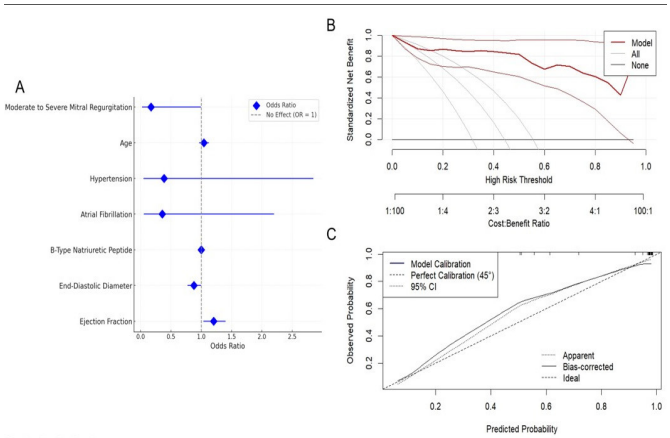

Figure 2. Predictive model for left atrial strain recovery in acute decompensated HFrEF (**A**) Forest plot displaying odds ratios for independent predictors of LASr recovery. (**B**) Decision curve analysis demonstrating the clinical utility of the predictive model across different risk thresholds. (**C**) Calibration plot assessing agreement between predicted and observed probabilities, indicating good model performance

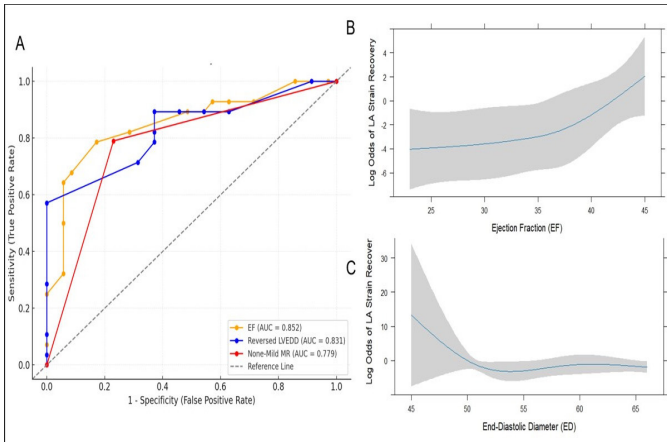

Figure 3. Echocardiographic predictors of LA strain recovery, (**A**) ROC curves for EF, reversed LVEDD, and none-mild MR. (**B**) Spline plot showing a positive association between EF and LA strain recovery. (**C**) Spline plot illustrating a non-linear relationship between EDD and recovery probability.

prognostic implications. Incorporating LASr recovery into routine echocardiographic assessment may aid in refining risk stratification and guiding management strategies in AHF, particularly in patients with significant MR or advanced cardiac remodeling. In addition, it may help support discharge decisions and identify patients who require closer follow-up, especially those with limited improvement despite decongestive therapy.

Limitations

This study has several limitations. First, the relatively small sample size may limit the generalizability of our findings. Second, LAS measurements were obtained using a single vendor's software, which may affect reproducibility across different platforms. Third, hemodynamic parameters, such as pulmonary artery wedge pressure, were not routinely assessed at discharge; instead, decisions were based on clinical stability rather than invasive measurements, potentially introducing variability in defining decongestion status. Moreover, due to the retrospective design, a certain degree of selection bias may exist, particularly related to the availability of adequate imaging and complete strain data, which may have resulted in the inclusion of relatively stable patients and thus could limit the applicability of our findings to the broader HF population. Additionally, factors such as neurohormonal activation and myocardial fibrosis, which could influence atrial function, were not evaluated. Lastly, the lack of long-term follow-up precludes determining whether LASr recovery translates into improved clinical outcomes.

CONCLUSION

As a result, this study is the first to investigate the determinants of LA strain recovery in acute decompensated HFrEF. Our findings highlight that LASr improvement during hospitalization is closely linked to baseline LV function, ventricular dimensions, and MR severity, suggesting that it

reflects not only volume status but also underlying cardiac structure. These results provide new insights into LA mechanics in HF and lay the foundation for future research to determine whether LASr recovery can serve as a prognostic marker or guide therapeutic strategies in this population.

ETHICAL DECLARATIONS

Ethics Committee Approval
This study was initiated with the approval of the Clinical Researches Ethics Committee of Başakşehir Çam and Sakura City Hospital (Date: 27.04.2022, Decision No: 135).

Informed Consent
Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process
Externally peer-reviewed.

Conflict of Interest Statement
The authors have no conflicts of interest to declare.

Financial Disclosure
The authors declared that this study has received no financial support.

Author Contributions
All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version

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